

Editorial

The case for early intervention in anorexia nervosa: theoretical exploration of maintaining factors

Janet Treasure and Gerald Russell

**Summary**

Here we revisit and reinterpret the original study in which the so-called 'Maudsley (London) model' of family therapy was compared with individual therapy for anorexia nervosa. Family therapy was more effective in adolescents with a short duration of illness. However, this is only part of the story. A later study describing the 5-year outcome contains important information. Those adolescents randomised to family therapy achieved a better outcome 5 years later. Moreover, the group with an onset in adolescence but who

had been ill for over 3 years had a poor response to both family and individual therapy, suggesting that unless effective treatment is given within the first 3 years of illness onset, the outcome is poor. We examine other evidence supporting this conclusion and consider the developmental and neurobiological factors that can account for this.

Declaration of interest

None.

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The course of anorexia nervosa

In addition to a raised mortality in anorexia nervosa there is often major physical and psychosocial disability. For example, a 9- to 14-year follow-up showed that over 20% of patients were unable to support themselves independently.¹ A similar outcome with over a quarter of patients continuing to have a poor psychosocial outcome at age 35 was found in a cohort of early-onset cases ascertained from community screening.² Thus the natural course even of mild, early-onset cases of anorexia nervosa can be protracted.

The duration of illness as a prognostic factor

The outcome of anorexia nervosa is predicted by body mass index (BMI), physical risk, age and illness duration. Recovery from anorexia nervosa becomes much less likely the longer the illness has persisted.³ Thus treatment for anorexia nervosa is more likely to be successful if the illness is recognised early – before weight loss becomes too severe and protracted. The most robust data to support this come from the family therapy trial conducted in the 1980s at the Maudsley.⁴ In this study, patients who had received preliminary in-patient treatment resulting in a return to normal weight were randomised to either family or individual therapy. Randomisation was stratified according to pre-determined outcome criteria, into two groups of patients with early-onset anorexia nervosa: (a) an early-onset short-history group, equivalent to an 'early intervention' (mean duration of illness 1.2 years); and (b) an early-onset long-history group, equivalent to a 'late intervention' (mean duration of illness 5.9 years). Outcomes were measured at the end of 1 year's treatment and 5 years after the end of treatment.⁵

At 1-year follow-up, the key measure was body weight, which showed a significantly higher gain in patients given family therapy early in the course of their illness. At 5-year follow-up the broader criteria of the Morgan & Russell scales were more appropriate and suggested that the family therapy had enduring superior effects. If the illness had an early onset but a long duration (over 3 years), it transpired that the family treatment was no more effective than the individual therapy and both were associated with a poor outcome. This demonstrates that an effective treatment (family therapy) applied early in the course of the illness can shape the outcome for at least 5 years.

Reappraisal of the Maudsley model

The National Institute for Health and Clinical Excellence (NICE) has only partly endorsed (a Grade B recommendation) the evidence for family therapy in anorexia nervosa.⁶ But since 2004 there have been further randomised controlled trials (RCTs) of family therapy which have been included in a 2010 Cochrane review.⁷ The authors used a random effects meta-analysis of 13 RCTs of family-based therapy, or variants. They concluded that the 1987 Russell *et al* study⁴ provided some evidence that family therapy may be more effective than individual supportive therapy in patients with a shorter duration of illness, in terms of remission, cognitive distortion and weight.

A generally good outcome of family therapy given early in the course of the illness has also been reported in a US trial where at a 4-year follow-up the mean BMI was 20.6 kg/m² and 90% of the patients had resumed menstruation.⁸

Since the Cochrane review, a two-site RCT (Stanford and Chicago) provides further evidence for the efficacy of family therapy.⁹ The comparison was between family-based treatment and adolescent-focused individual therapy in an impressively large group of adolescent patients ($n = 121$) aged 12–18, with a mean duration of illness of 11.3 months. Assessments were made at the end of 1 year's treatment, and at 6-month and 12-month follow-up post-treatment. Both treatments led to marked improvement with similar full remission rates at the end of treatment. However, family-based therapy was superior in causing full remission rates at both follow-up points.

Thus there is now substantial support for the original Maudsley findings that family therapy produces a good long-term outcome in young patients with a short duration of illness. But what about the other finding from the Maudsley study, which suggested that if the illness had persisted for longer than 3 years, the 5-year recovery rate was low? The authors of the Cochrane systematic review⁷ considered this to be an interesting hypothesis and recommended further studies designed to distinguish the impact of chronicity from age.

What causes inadequately treated anorexia nervosa to persist?

The obvious answer is starvation and stress. The nutritional consequences of eating disorder behaviours on the brain itself have been somewhat overlooked and yet the brain is particularly vulnerable. First, anorexia nervosa develops at a time when the brain is undergoing structural (dendrite pruning and myelination) and functional changes. Second, the brain utilises about 20% of the total calorie intake and is particularly dependent on glucose, leading to the dysfunctional effects of extreme caloric restriction. Third, the brain plays a major role in the control of eating, through the neural circuitry regulating the drive for food, and this can be perturbed by eating disorder behaviours.

The following review is admittedly speculative but should encourage new research approaches.

Developmental changes

Brain development undergoes significant modification throughout adolescence. As well as streamlining connectivity, hormonal changes have an impact on the development of the 'social brain'.¹⁰ Maturation of the prefrontal areas which exert self-regulatory control is later than that of the subcortical areas. Thus the balance between reflection, risk taking and impulsive behaviours is in a state of flux. These transitions in brain organisation may contribute to the risk of developing an eating disorder in adolescents. Moreover, it is highly likely that poor nutrition, hormonal changes and high levels of stress are disruptive to brain maturation. This effect may make it harder to recover from anorexia nervosa.

Nutrition and brain structure and function

During the starvation and general upheaval of acute anorexia nervosa brain mass and brain function are reduced, particularly self-regulatory systems.¹¹ For example, executive control is impaired with problems in set-shifting, attention and decision-making. Subcortical regions such as the amygdala and the basal ganglia are free from 'top-down' control. Thus there is a failure to inhibit compulsive intrusions and to regulate emotions. The subcortical (implicit) system is biased towards sensitivity to punishment with indecision and compulsivity. The resulting motivational inhibition and compulsive behaviour serve to maintain eating disorder behaviours.

Moderation of reward systems in the brain

The impact that eating disorder behaviours themselves have on the brain and the control of appetite is exemplified by models of binge eating in laboratory rodents. Scientists have produced experimental 'food addiction' by replicating in the laboratory the conditions for inducing binge eating, such as food restriction, gastric drainage (an analogue of vomiting), stress and intermittent

access to highly palatable food. Not only do these animals 'binge' eat but they also show a propensity to relapse after a time and cross-tolerance to alcohol and cocaine. Similar neuroadaptations follow exposure both to food and drug rewards and the motivation to attain these rewards is altered.¹¹ Indeed, the attentional bias to food in eating disorders mirrors the processes activated by drug cues in addictions (for a review see Treasure *et al*¹²). This sensitisation of brain reward systems may explain the switch from extreme restriction to bulimia nervosa, a closely linked variant of anorexia nervosa.

Conclusions

The first RCT of family therapy highlights the detrimental effects of prolonged untreated anorexia. We have illustrated how anorexia nervosa can interfere with brain function and how this can make the disorder more resistant to treatment.

What lessons can be learnt from this? Early recognition and intervention are vitally important and family therapy can play a special part in the young. Since the original Maudsley trial, family therapy has become diversified, including conjoint versus separated types and multifamily interventions. The essence of family therapy, the Maudsley model, consists of mobilising parental resources by:

- (a) exonerating parents from causing the illness;
- (b) getting them to take joint control of their child's eating until the child becomes able to keep his or her body weight at a normal level.

There remain obstacles to be overcome as it is the rule that people in the early phase of anorexia nervosa do not accept that there is anything wrong. Ways need to be found to engage resistant patients such as working within their social network and overcoming the barriers to help-seeking within the health services. Early recognition in schools and family support may reduce delay. Correcting malnutrition and the secondary consequences of brain starvation are essential in parallel with psychosocial help.

The complex needs of people with anorexia nervosa mirror those of people with psychosis in that there is a need to minimise the time of untreated symptoms early in the illness;¹³ those with a severe enduring form of illness need help with the factors that maintain the illness. Following the psychosis example, services may need to be reconfigured to match these phases of the illness with an early intervention service which bridges the age gap from 14 to 35 years and a recovery rehabilitation service including some assertive outreach for those who are hard to engage.

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poems
by
doctors

Scored

Peter Wells

Eyes, as old as autumn leaves
on the death mask of a child,
marble cold.

Boot black hair
carefully unkempt.

A stalk,
scarred with needle tracks
cupped beneath her witch face
still avid for spells.

But I'm no sorcerer
and heroin is not within my gift.

'Piss off then!' she cried
with a cripple's touchy pride,
her trouser pocket poked out
like a tongue,
the valedictory gesture of the damned.

Peter retired in 1993. He opened a young people's unit in 1970 for the treatment of disturbed adolescents in the North region. He began writing poetry while serving in the Royal Navy in 1943.

Another of Dr Wells's poems was published in the November 2010 issue of the *Journal*.

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